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ABSTRACT

Designed as a public information pamphlet, the text discusses the problem of lead poisoning in children. The preventable nature of the problem is stressed as well as needed action on the part of the public, physicians and other health workers, and the legislators. The pamphlet emphasizes that each of these areas is essential in preventing death or disabilities to children in urban areas. (CD)

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FOREWORD

Lead poisoning is a needless cause of mental retardation, other neurological handicaps and death among children.

LEAD POISONING IN CHILDREN stresses the preventable nature of this health problem and is intended to stimulate some much needed action on several fronts:

- The public must be made aware that lead poisoning is preventable, be able to recognize its symptoms and seek appropriate medical help, and cooperate in efforts to eliminate this health problem.

- Physicians and other health workers need more information than many now have about the availability of screening, diagnostic, and treatment methods.

- Legislative action is needed to see that lead paint is removed from dilapidated housing and that proper warnings are given in the use of any paint containing lead.

LEAD POISONING IN CHILDREN emphasizes that each of these steps is essential if progress is to be made in preventing needless death or handicapping to children living in our urban areas.

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lead poisoning in children

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lead poisoning in children

Lead poisoning in children, resulting mostly from ingestion of chips of lead-containing paint from walls and woodwork in old, dilapidated housing, remains a unique public health problem. Its etiology, pathogenesis, pathophysiology, and epidemiology are known. Practical methods are available for screening, diagnosis, prevention, and treatment. Yet each year lead poisoning continues to cause the deaths of many children and mental retardation or other neurological handicaps in many other children.

Health workers should be reminded, and the public informed, that lead poisoning is preventable. As is true with many other diseases, total prevention may be difficult to achieve, but significant reduction in the number and severity of lead poisoning cases can be expected from a well-planned program.

The following is an analysis of many facets of lead poisoning in children based on a review of the literature. A plan of approach to this health problem is suggested.

Size of the Problem

Lead poisoning in children is not an uncommon occurrence in the United States. Although slum areas in large old cities appear to have by far the greatest incidence, this problem is not necessarily restricted to the poor; it has been reported in children from economically and socially advantaged homes (*29*).

It is difficult if not impossible to assess the true incidence of lead poisoning in children. Obviously, in any community where overt lead poisoning associated with

pica is reported, many unrecognized, subclinical cases must also exist. But while one cannot talk in terms of incidence rates, from the number of cases reported in several large cities one must conclude that this health problem is quite common in many areas.

In New York City, over 500 confirmed cases of lead poisoning in children were reported in 1964. This is excluding over 100 suspected cases that were being observed or investigated. Of 61,167 poisonings reported to the New York City Poison Control Center between 1955 and 1963, 3 percent or 1,704 were cases of lead poisoning (*19*). In Baltimore, during 1956–1964, there were 1,337 known cases of lead poisoning in children (*32*). In Chicago, during 1959–1961, 429 cases of lead poisoning were reported to the board of health. They represented 4.7 percent of the cases of accidental poisoning reported in that period (*9*).

Since these reported cases merely represent a portion of the total extent of lead poisoning, perhaps the following survey data are a better indication of the actual prevalence of this problem in slum areas.

In Cleveland, a survey was conducted among 549 children aged 12–35 months living in areas of old, poorly maintained housing where flaking paint was frequently found. Of these children, 28 percent had an abnormal urine that might be indicative of increased exposure to lead, and 6.4 percent fulfilled the diagnostic criteria for lead poisoning. Of 105 children of similar socioeconomic background living in a new housing project, none had significant evidence of lead poisoning (*17*). In Baltimore, among 604 children aged 7–60 months who came from a low-income congested area where lead poisoning was known to have occurred, 333 had clinical or laboratory evidence or a history suggestive of increased exposure to lead. Of these 333, 148 had blood lead levels exceeding 0.05 mg./100 ml. (*4*). Survey of a suspected high incidence area in Chicago disclosed that out of 500 study patients,

7.9 percent had clinical or laboratory evidence compatible with the diagnosis of lead poisoning (*9*).

Consequences of Lead Poisoning

Mortality

In a 3-year period, 1959–1961, lead poisoning accounted for 4.7 percent of 9,853 cases of accidental poisoning in children reported to the Chicago Board of Health, but it was responsible for 79 percent of the total deaths due to accidental poisoning for the same period (*9*).

Between 1959 and 1963, 182 children were treated for acute lead encephalopathy at Cook County Children's Hospital. Over the 5-year period, despite the use of chelating agents and various techniques for reducing intracranial pressure, the case fatality rate remained essentially unchanged at the 25 percent level (except for one year when a higher fatality rate occurred, reportedly as a result of the bilateral craniectomies employed as part of the treatment that year) (*15*). In Cleveland, the mortality rate reported for lead poisoning from 1952 through 1958 was 30 percent (*17*). Coffin et al. recently reported a mortality of 4.5 percent in a group of 22 children with lead encephalopathy who were treated with a combination of BAL (British anti-lewisite) and CaEDTA (calcium disodium versenate) and measures to control cerebral edema (*10*).

Morbidity

For many of those who survive, the outlook remains grim. In Chicago, a study of 425 children who were fol-

lowed for 6 months to 10 years after treatment for lead [illegible] logical sequelae. Among the 59 children in this group who had presented encephalopathic symptoms initially, 82 percent were left with handicaps: 54 percent had recurrent seizures, 38 percent were mentally retarded, 13 percent had cerebral palsy, and 6 percent were found to have optic atrophy. Some had multiple handicaps (*30*). Other sequelae reported in children who had lead encephalopathy include behavior problems, inadequate interpersonal relationships and inability to comprehend the abstract (*5*, *28*). Lead poisoning appears to be a cause of renal impairment, according to extensive epidemiological surveys conducted in Australia.

The exact incidence of lead poisoning as a cause of mental retardation is not known, but limited surveys of blood lead levels among mentally retarded children suggest that the incidence is probably not infrequent (*3*, *29*, *37*). On the other hand, it has been contended that mentally retarded children are more likely to have pica and therefore more likely to have lead poisoning (*3*).

Several followup studies have indicated impairment of intellectual ability in children who had lead poisoning. In the series of 425 children with lead poisoning reported from Chicago by Perlstein et al., mental retardation was found to be the most frequent sequela, occurring in 22 percent of the children. Among those who presented symptoms of lead encephalopathy initially, 38 percent of the children were found to be mentally retarded at the followup study, as mentioned above (*30*).

Similar results have been reported by other investigators. Byers and Lord followed 20 children who had relatively mild lead poisoning and were discharged from hospitals as recovered. They found that the IQ's of these children 3 to 12 years later ranged from 67–107, with a mean of 90. All but one of these children showed unsatis-

factory progress in school because of specific intellectual defect (*a*, *b*). Jenkins and Mellins studied 32 children who had severe lead poisoning (nearly all had evidence of encephalopathy) and found that 6 to 8 months later, their IQ's ranged from 35–115, with a mean of 74. The majority were severely retarded (*23*). Smith reported that five or more years after lead poisoning, a group of children who had lead encephalopathy had an average IQ of 80 (range 58–104), while those who had lead poisoning without encephalopathy had an average IQ of 87 (range 75–117); a group of controls who had pica without lead poisoning had an average IQ of 98 (*33*).

Epidemiology of Lead Poisoning

1. "High risk" areas for lead poisoning are almost synonymous with the slums, where old, deteriorating housing prevails. In these areas, accessibility to flaking paint and broken plaster, high incidence of pica, and lack of adequate parental supervision provide an optimum environment for lead poisoning (*20*).

2. Children between the ages of 1 and 6 years are the main victims; those between 1 and 3 years of age comprise approximately 85 percent of the cases, with the highest incidence at age 2 years (*20*, *28*). Over 50 percent of all deaths from lead poisoning occur in 2-year-olds (*19*).

3. Childhood lead poisoning is significantly related to pica. In New York City it has been reported that over 30 percent of children who manifest pica have lead poisoning (*19*). Seventy to ninety percent of children with lead

poisoning have been found to have a history of pica (*4*, *19*, *22*).

4. Symptomatic lead poisoning in children has a definite seasonal variation. About 45 percent of the cases reported in New York City during 1954–1963 occurred in the summer months of June-September (*19*, *22*). Others report that 80–85 percent of the cases occur in these months (*9*). But lead poisoning should not be considered a summertime disease. More and more cases are being reported in the winter months as health workers become increasingly aware of this problem. Some cases occur in winter when leaded battery casings are burned for fuel and the fumes are inhaled or there is prolonged contact with the ashes (*6*, *35*). Epidemiologic studies indicate that lead encephalopathy is much more frequent during the summer, but asymptomatic lead poisoning is a year-round disease (*19*).

5. There is a high incidence among Negroes and Puerto Ricans, probably because a greater proportion of these groups live in the so-called lead belts (*20*, *22*).

6. There is no significant difference in incidence by sex (*20*).

7. A high incidence occurs among siblings. McLaughlin reported 19 children with clinical lead poisoning from nine families; six of the children died (*27*). A 30 percent incidence among siblings has been cited by others (*14*).

8. There is a high recurrence rate (*27*, *33*, *35*).

9. Lead poisoning associated with pica is a chronic process. From 3 to 6 months of fairly steady lead ingestion is necessary in most cases before clinical manifestations develop (*11*, *35*).

Diagnosis and Screening

Diagnosis

Since, in its early stage, lead poisoning is often asymptomatic or merely manifested by symptoms commonly seen in association with other diseases in everyday pediatric practice, to those unfamiliar with lead poisoning the correct diagnosis may not even be suspected. Vague, nonspecific symptoms, such as anorexia, abdominal pain, constipation, vomiting, anemia and irritability, are usual in early cases. Often, parents do not volunteer any pertinent information. A survey of 300 confirmed cases of lead poisoning revealed that 76 percent of the children had no presenting complaints, but on specific inquiry it was found that 58 percent of them had anorexia and 9 percent had vomiting (*19*). Among the 22 children with severe lead encephalopathy studied by Coffin et al. (*10*) 18 had been treated symptomatically by local physicians for "gastroenteritis" for varying periods prior to the onset of central nervous system symptoms. In addition, some had been treated for anemia, constipation, glycosuria, gait disturbances and sudden onset of strabismus.

A history of lead ingestion, or the presence of suggestive signs and symptoms in a young child is very useful in substantiating a diagnosis of lead poisoning, but confirmation of the diagnosis requires the demonstration of increased amounts of lead in blood or urine. Blood lead determination is widely accepted as the most reliable and practical method of diagnosing lead poisoning in children, as urinary lead determination requires a 24-hour specimen, and the excretion of lead is influenced by fluid intake, renal function and other factors (*19*, *35*). Lead levels in blood should be interpreted with caution, since values are affected by factors such as hematocrit, intercurrent infection, coincident bone disease, or recent ad-

ministration of chelating agents (*8*). Caution should be taken to use lead-free equipment in collecting and storing blood samples. Laboratories may vary in accuracy; a physician should be aware of any general tendency in the laboratory he uses to give high or low test results for blood lead determinations.

Jacobziner suggested the following criteria for making a diagnosis of lead poisoning: a blood lead level of 0.06 mg./100 ml. or higher, and the presence of two or more of the following signs and symptoms: gastrointestinal symptoms of anorexia, vomiting, abdominal pain, or constipation; hematologic finding of anemia or pallor; neurologic signs of irritability, stupor, lethargy, or convulsions; and roentgenologic signs of increased density of the long bones, or opacities representing lead flakes in the abdomen. If a patient is asymptomatic but has a definite history of pica and a blood lead level of 0.06 mg./100 ml., he is classified as a "possible" case of lead poisoning, and periodic blood lead determinations are carried out (*19*).

Recently, Chisolm proposed that the "normal" blood lead level be revised downward. According to him, after infancy the median blood lead level is 0.027 mg./100 ml., and the upper normal limit should be set at 0.04 mg./100 ml. The widely used limits for normal of 0.05 and 0.06 mg./100 ml. were based on the use of samples containing a large proportion of young children from old urban housing areas who may have had increased exposure to lead (*8*).

The so-called classical signs of lead poisoning, i.e., lead lines on bone X-rays, radiopaque materials in the gastrointestinal tract, basophilic stippling of erythrocytes, and coproporphyrinuria, are often absent, especially in early cases and in children under 2 years. In one study, only 17 percent of patients with early lead poisoning and 45 percent of those with late poisoning had positive or borderline lead lines on X-rays. In four of eight children with lead encephalopathy, the X-rays were normal (*31*).

Basophilic stippling was reported to be present in 60 percent of childhood cases of lead poisoning by one author, and in only 30–40 percent of cases by another (*16*). Similarly, coproporphyrin III is sometimes absent in the urine of children with lead poisoning (*4*, *10*). In one survey, blood lead determination was positive (0.06 mg./100 ml. or higher) in 37 percent of the suspected cases, but urinary coproporphyrinuria of 2+ or higher was present in only 5.5 percent of the patients (*19*). These "typical" signs are therefore only useful if present, and their absence does not rule out the possibility of lead intoxication. It is obvious that if one were to wait for the classical signs to appear before making a diagnosis, many children would have progressed to the stage of irreversible neurological damage while others would be diagnosed only on autopsy tables.

Many workers in this field feel strongly that treatment should be begun on any child with clinical symptoms suggestive of lead poisoning or any child with abnormally high blood lead levels even if he is symptom-free. Treatment should not be delayed until a conclusive diagnosis is available (*8*).

Large-scale screening methods

At present, blood lead determination appears to be the most reliable test available for screening children for lead poisoning.

Urinary coproporphyrin determination has been used by some health workers, but its value as a screening test has been questioned by others (*11*, *13*). While the usual technique is relatively simple, positive results may be indicative of conditions other than lead intoxication, and negative results are frequently encountered in the presence of lead poisoning. Benson and Chisolm have devised a urinary

coproporphyrin test which they have found to be uniformly positive (3+ or 4+) in patients with whole blood lead concentration greater than 0.10 mg. per 100 gm. (*2*). The test is less useful for detecting children with lower elevations of blood lead level.

Urinary excretion of delta aminolevulinic acid (ALA) is increased in lead poisoning, but this metabolite appears to be a sensitive indicator of lead toxicity rather than of early increased lead exposure. Furthermore, urinary ALA is related to urinary concentration and can vary considerably within a 24-hour period. Testing of random urine samples for this metabolite is therefore unreliable. A simplified urinary ALA test was described by Davis, et al., to be a rapid and useful test for screening children for lead poisoning (*13*). However, his results could not be duplicated by investigations conducted in New York City and Chicago. In these cities, an unduly high percentage of false positive and false negative results was reported with this test. The collection of urine samples from young children who are not toilet trained but among whom the incidence of lead poisoning is highest also poses a definite problem.

Hair, a continuously growing tissue which provides a metabolically passive and irreversible pathway for lead, is known to concentrate more lead per unit weight than any other tissue or body fluid, including bone, blood, and urine. Recently Kopito, Byers, and Shwachman studied the lead content of hair of 16 children with confirmed diagnosis of chronic lead poisoning (*24*). With one exception all had either elevated concentration of lead in their hair or significantly higher values in the segments proximal to the scalp in comparison with the distal segments. The

mean lead concentration in hair of the 16 children with lead poisoning was 282 μg./gm., while that of 41 control children with 24 μg./gm., a difference of high statistical significance. In eight children, the concentration of lead in the proximal segment was 1.5–3.8 times higher than in the distal segment. The procedure requires only 10 mg. of hair in 5 to 10 mm. segments. The ready availability of hair as a specimen, the ease with which it can be collected, sorted and transported, and the reasonably simple technique of this procedure are the salient features which favor it as a screening device for lead poisoning in children. Further corroboration is essential, however, before this procedure can be accepted as a reliable screening test.

Two other tests reported to be valuable for screening of this condition are the determination of fluorescence of erythrocytes (fluorescytes) (*36*) and ophthalmoscopic examination for retinal stippling (*34*). These tests also require further evaluation.

Factors Contributing to Lead Poisoning

Although lead can be absorbed into the body via various routes such as inhalation and skin absorption, in children lead poisoning results almost exclusively from ingestion of flaking and peeling lead-containing paints found in old houses and on old furniture. Some of the most important factors which work together to perpetuate lead poisoning in children include:

Dilapidated housing

In the large, old cities, there is a marked concentration of lead poisoning cases in slum areas—the so-called

lead belts. Here, dwellings often have several coats of paint on walls, woodwork, and ceilings, and the base coats generally contain significant amounts of lead. Until about 1940, lead-containing paint was frequently used in interiors as well as exteriors of houses. The houses are usually in bad repair and paint peelings and loosened plaster provide a dangerous source of lead to children with pica. Thus, despite legislative effort to prohibit the use of lead-containing paint for interiors, lead poisoning continues to occur in children living in slums.

A 1958 survey of 100 dwellings occupied by Puerto Ricans in Philadephia revealed that 87 percent had at least one room in which the lead content of the painted surface was above 1 percent, the maximum level considered safe (*18*). In 1957, a survey of 100 blocks of dwellings in Baltimore randomly selected for lead paint sampling disclosed that 70 percent of 667 dwelling units had paint containing lead in excess of 1 percent (*32*). In contrast, lead poisoning was not found among children living in newly constructed housing projects in New York City, even in the so-called high incidence districts. This indicates that lead poisoning in children is inextricably linked to old, dilapidated housing (*19*).

Lack of awareness about the problem among physicians and other health workers

Many physicians are not aware of the existence or the magnitude of this problem of lead poisoning, either because they seldom encounter cases of it in their practice or because the cases, when encountered, are not correctly diagnosed. In New York City, where several hundred confirmed cases of lead poisoning are reported annually, not a single case was reported to have been seen at a large medical center over a 3-year period (*1*).

Some equate the manufacture of lead-free paint today with the extinction of lead poisoning in children. What they do not realize is that old houses often still contain many layers of lead-containing paints, that today paints manufactured for outdoor use still contain lead, and that people unaware of the hazard of lead may use the outdoor paint for interior purposes.

An illustration of the misconceptions some physicians have concerning lead poisoning in children is the case of an 18-month-old boy who was admitted to the Massachusetts General Hospital with lead encephalopathy. The child was known to have pica, and, a few months prior to admission, he was seen by two physicians on separate occasions. One physician did not consider the possibility of lead poisoning because he had the common misconception that paint ingestion is harmless since interior paints manufactured today do not contain lead. The other erroneously informed the mother that drinking large amounts of milk will prevent lead poisoning due to paint ingestion (*14*).

Even among those who are aware of the problem of lead poisoning, some are hesitant to make a positive diagnosis. A large number of cases have been reported by hospitals to the Poison Control Center of New York City as "possible lead poisoning" despite highly suggestive clinical symptoms and blood lead levels considerably above the standard used for positive diagnosis (0.06 mg./100 ml.). One child admitted to a hospital with convulsions and vomiting had a blood lead level of 0.32 mg./100 ml. Although this was more than five times the level accepted for diagnosis, the child was reported as a case of "possible lead poisoning" (*21*). Hesitancy in making a diagnosis often leads to undue delay in treatment, and the loss of invaluable time during which irreparable damage may occur.

A poorly informed public

Many parents are not aware of the danger associated with pica or the consequence of paint ingestion. Jacobziner reported that, in 90 percent of the cases of lead poisoning in New York City, the family knew that the child was ingesting paint but were unaware that this practice was hazardous (*19*). Another example of lead poisoning directly related to the lack of public information about the sources of lead intoxication is the sporadic occurrence of large scale poisoning from inhalation of lead fumes produced by burning wooden battery casings impregnated with lead salts for fuel (*8, 35*). Lead poisoning associated with ingestion of home-grown vegetables produced on a soil containing numerous lead battery casings has also been reported (*29*).

Inadequate prevention of reexposure to lead

There is a high rate of recurrence of lead poisoning among children. In a series reported by Smith, 19 percent of 229 cases of lead poisoning in Cincinnati had recurrent episodes (*33*). McLaughlin of New York City reported a total of 151 admissions of 143 children over a 5-year period. Several children were admitted two and three times, each time with recurrent encephalopathy. In one case, repeated episodes left the child completely incapacitated (*27*). Others working in the field of lead poisoning have also stated that "we saw the same children over and over again being brought in for more deleading, and each time with evidence of more encephalopathy, more residual brain damage. We were seeing mental retardates and institutional vegetables created right under our eyes" (*11*).

It is thus apparent that failure to prevent re-exposure to lead contributes significantly to the mortality and morbidity of lead poisoning. Chisolm and Harrison have emphasized that the severity of residual handicaps may be correlated with the duration of exposure to lead and with the incidence of recurrent episodes (*7*).

An Approach to Control and Prevention

Knowledge of the epidemiologic data and factors contributing to lead poisoning in children can be translated into programs directed at the control and prevention of this health hazard. The effectiveness of such programs is best illustrated by the results obtained in New York City. After programs were set up for early diagnosis and treatment, the number of children with lead poisoning reported annually rose from an average of 29 during 1950–1954 to over 500 in 1964. Simultaneously, the fatality rate dropped from 27 percent (1950–1954) to 1.4 percent in 1964 (*19*).

In the following plan of approach to control and prevention, action is suggested in several areas: professional and public education; casefinding; followup of cases; legislative measures; research; and improved housing.

Educational campaign

Directed to physicians and other health workers. The encouraging results in New York City were attributed to the early casefinding techniques, and increasing the phy-

sician's awareness of lead poisoning was a major factor in early casefinding, according to Jacobziner (*20*). Educational programs for the medical profession, nurses and other health workers in the form of talks, exhibits, and distribution of literature can be conducted in medical and nursing schools, hospital conferences, and professional meetings and conventions. Physicians must be made aware that lead poisoning is still a health problem today. They must learn to make routine inquiry about pica in children 1–6 years of age. They must also learn to think of lead poisoning when confronted with children who have symptoms compatible with that diagnosis. They must become familiar with the methods of screening and diagnosis, and know what facilities and services are available locally for making a diagnosis (such as blood lead level determination by the health department).

Directed to the public. The public and, in particular, parents with young children, should be informed of the hazards of lead, the sources of lead (old paints, plaster, storage battery casings, gasoline, etc.), the methods by which lead poisoning can occur (ingestion, inhalation, etc.), and the danger in pica. They should learn to recognize the early symptoms of lead poisoning, and be instructed to seek medical help when such poisoning is suspected.

Casefinding program

Screening in clinics, hospitals, and health projects (e.g., Children and Youth Projects supported by Maternal and Child Health Service, the Head Start projects):

1. Routine inquiry about pica in all children 6 years old or younger.

2. Blood lead level determination in all children with a history of pica, and in siblings of children with either positive lead poisoning or pica.

3. Reevaluation at regular intervals of children with pica who at initial screening did not have toxic blood lead levels. Children in "high risk" areas who have pica are in constant danger of lead poisoning, and an initial negative screening test does not guarantee that they may not be poisoned later on.

4. Screening of all children aged 1–6 years from high risk areas by blood lead determination where resources permit.

Home surveys. Home visits are especially useful when parents of children with lead poisoning fail to bring siblings in for screening. Similarly, visits to families living in a housing project where lead poisoning has occurred will often uncover other cases.

Provision of prompt service in blood lead determination by local health departments. The health department of New York City reports results of blood lead determination to physicians and hospitals within 24 hours after receipt of the specimen (*20*). In other places, it may take much longer (*11*). In many cases, delay in diagnosis leads to delay in treatment, which in turn increases the risk of encephalopathy and irreversible damage in the patient.

Followup program

Prevention of reexposure to lead. Since lead poisoning usually occurs among those least able to improve their environment, prevention of reexposure will in many cases require formidable efforts by health and social workers:

1. Instruction of the parents when a child is discharged from the hospital or clinic after treatment, regarding the seriousness of repeated exposure to lead.

2. Home visits by health workers to determine whether exposure to lead is continuing and to help the family prevent further exposure.

3. Removal of lead from environment. Paint removal is an expensive, time-consuming process, but it should nevertheless be required of the landlord when it is necessary for prevention of reexposure. The child should not be allowed in the home while lead paint is being removed, since there would be great danger of further exposure.

4. In some cities and hospitals, whenever removal of lead from a home is not immediately feasible, the child is placed in a convalescent or foster home until his home has been made safe or the parents have found better housing. This measure may not be practicable in many places because of the lack of suitable foster homes, but it should certainly be considered whenever the only alternative is to send a child home to a dangerous situation.

5. Additional social casework, if indicated, to reduce psychological or cultural factors resulting in pica in the child.

Determination of blood lead levels at regular intervals. This should be done in cases where prevention of reexposure to lead has failed.

Legislation

In those jurisdictions, particularly those including large urban areas, where the following measures are not

in effect, legislative action ought to be considered:

1. Reporting by physicians of lead poisoning cases to local health departments.

2. Ordinances requiring that dwelling places be maintained in good repair and fit for human habitation, and that any condition found to be dangerous or detrimental to life or health (such as flaking lead-containing paint and loose plaster coated with such paint) be removed.

3. Prohibition of the use of paints containing lead for indoor purposes and on toys and furniture.

4. Warning labels on all paints containing more than 1 percent lead.

Further research

Causes and treatment of pica. Pica is a serious problem among children. In the Children's Hospital of the District of Columbia it was reported to occur in 50–60 percent of all children 1–2 years old in the Negro clinic population. Among the white private patients, the incidence was still surprisingly high—28 percent of the 1- to 2-year olds (*11*). Pica is responsible not only for lead poisoning, but also for other types of accidental poisoning in children. In the Children's Hospital of the District of Columbia it was found that in children admitted because of ingestion of poisons, 67 percent of those in the early age group had pica (*11*).

Various psychological and cultural factors have been cited as contributing to the development of pica. Improper child-rearing practices in particular have been said to result in needs in the child which he satisfies through pica (*25*). Further research into the causes and treatment

of pica is a logical step in combating accidental poisoning in childhood.

Laboratory screening procedure for lead poisoning. Blood lead determination, even with its pitfalls, is generally considered to be the most reliable of the many indices or biological tests of lead exposure and absorption. However, the available methods of blood lead determination which are applicable on a large scale generally require at least 5 ml. of blood obtained by venipuncture. A micromethod is being developed and hopefully will be available before too long.

Treatment of lead encephalopathy. The report of Coffin et al. indicates that improved methods of treatment can effect a marked reduction in the mortality associated with lead encephalopathy. But in the series of cases reported the residual morbidity caused by lead encephalopathy remained high. Seven of the twenty-two children treated were left with residual injuries. Although the use of both BAL and CaEDTA removed lead from the body and brain cells more effectively than when either agent was used alone, the control of cerebral edema remained a difficult problem. Various therapeutic agents, including urea, mannitol, and dexamethasone, and surgical decompression have been used to combat this problem, but none has proved to be completely satisfactory. Continued research to reduce both the mortality and morbidity from lead encephalopathy is urgently needed.

Slum clearance

Among the benefits of clearing cities of old, dilapidated housing is the reduction of lead poisoning in children due to paint ingestion. This point should not be over-

looked in the proposals of citizens and government leaders for slum clearance and for improved low-cost housing. Because these goals in housing are far from being achieved in most large cities, however, they cannot be relied upon as the main routes by which lead poisoning will be prevented in today's young children. Educational campaigns, casefinding, followup, and other programs must be carried on vigorously as specific measures against lead poisoning in children.

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